Potassium protects the heart

Genetically salt-sensitive rats were fed a normal diet (0.3% sodium chloride) or a high-salt diet (8% sodium chloride), with or without high potassium intake (8% potassium chloride) for eight weeks. High salt intake significantly increased blood pressure, impaired left ventricular relaxation, and increased the activity of NADPH oxidase in cardiac tissue. High potassium intake improved left ventricular relaxation and reversed the elevation of NADPH oxidase activity, even though blood pressure remained high.

In other studies, dogs made potassium-deficient by means of a low-potassium diet and administration thiazide diuretics showed impairments of both systolic and diastolic heart function, with the most striking effect being a 49% reduction in the peak rate of ventricular filling. In a study of healthy human volunteers, potassium depletion resulted in a 14% reduction in the peak rate of ventricular filling. In a randomized trial, elderly men who switched from regular salt to potassium-enriched salt (49% potassium chloride, 49% sodium chloride, 2% other additives) for 31 months had a 41% decrease in cardiovascular mortality.

Comment: Diastolic dysfunction (impaired left ventricular filling) is a common and important, though under-appreciated, cause of heart failure. In one recent study, of 556 unselected patients with heart failure, 55% had a normal left ventricular ejection fraction, indicating that heart failure was almost certainly due to diastolic dysfunction. Patients with diastolic heart failure have mortality rates similar to those with systolic heart failure.

The studies reviewed above suggest that potassium deficiency can contribute to diastolic dysfunction and possibly to systolic dysfunction as well. One mechanism by which potassium might help prevent heart failure is by inhibiting cardiac NADPH oxidase activity, thereby decreasing oxidative stress.

Most people do not consume enough fruits and vegetables (the main dietary sources of potassium). Some diuretics used to treat heart failure may also promote or exacerbate potassium deficiency. In addition, some of the pathophysiological mechanisms underlying heart failure may lead to a loss of both potassium and magnesium from myocardial cells. Magnesium is required for the intracellular uptake of potassium, so potassium supplementation alone will not correct intracellular potassium deficiency unless magnesium deficiency is also corrected. In patients with renal failure (which is sometimes associated with heart failure), potassium and magnesium should be administered with caution and monitored with appropriate lab tests.


Improving cardiac risk factor with a nutritional supplement

Forty men with a history of myocardial infarction who had completed a cardiac rehabilitation program (including supervised exercise training and lifestyle and dietary recommendations) were randomly assigned to receive 500 ml/day of a fortified dairy product (active-treatment group) or 500 ml/day of semi-skimmed milk (control group). The fortified product provided the following nutrients that were not present in the control drink (alpha-linolenic acid, 0.6 g/100 g of total fat; eicosapentaenoic acid, 2.1 g/100 g of fat; docosahexaenoic acid, 1.2 g/100 g of fat; vitamin E, 15 IU/L; vitamin B6, 3 mg/L; and folic acid, 300 mcg/L). The
fortified product also contained larger amounts of oleic acid than the control drink. The mean C-reactive protein concentration decreased from 3.90 mg/L at baseline to 2.01 mg/L after 12 months in the active-treatment group and from 3.64 mg/L to 3.23 mg/L in the control group (p < 0.05 for the difference in the change between groups). The difference between groups was also significant after six months.

Comment: C-reactive protein is a marker of inflammation. An elevated serum C-reactive protein level is an independent risk factor for coronary heart disease. The results of this study demonstrate that supplementation with modest doses of fatty acids, vitamin E, and B vitamins can significantly reduce C-reactive protein levels in patients with a history of myocardial infarction. Another way to reduce C-reactive protein levels is to avoid heavily cooked foods (such as well-done meat and pizza) and to emphasize raw or lightly cooked foods. Byproducts formed during harsh cooking (advanced glycation end products) appear to promote inflammation. For people who are overweight, weight loss also reduces C-reactive protein levels.

Does vitamin D deficiency lead to heart failure?

This review article points out that the majority of patients with congestive heart failure have vitamin D insufficiency (serum 25-hydroxyvitamin D < 50 nmol/L or < 20 ng/ml). Vitamin D deficiency in these patients is presumably due in part to relatively low amounts of outdoor activity. Vitamin D deficiency may contribute to the pathogenesis of heart failure, or may exacerbate heart failure, by promoting the development of hypertension or by increasing parathyroid hormone levels, which is believed to contribute to the development of cardiovascular disease.

Comment: In recent years, evidence has accumulated that vitamin D deficiency is very common and may contribute to the development of muscle weakness and pain, some autoimmune diseases, osteoporosis, infections, and cancer. While vitamin D deficiency has not been proven to cause or exacerbate heart failure, it would be worthwhile to measure serum 25-hydroxyvitamin D levels in patients with heart failure and to correct deficiencies either with supplementation or sunlight exposure. The amount of oral vitamin D required to optimize 25-hydroxyvitamin D levels might be higher in patients with heart failure than in healthy people, because heart failure is often accompanied by bowel wall edema, which could inhibit nutrient absorption.


Don't forget thiamine for heart failure patients

The prevalence of thiamine deficiency (as determined by erythrocyte thiamine pyrophosphate concentrations) was significantly higher in 100 patients with congestive heart failure than in healthy controls (33% vs. 12%; p = 0.007).

Comment: Patients with congestive heart failure may be at increased risk for thiamine deficiency as a result of diuretic-induced thiamine excretion, malnutrition, and advanced age. Severe thiamine deficiency is a known cause of heart failure (beri beri heart disease), and it is likely that even modest degrees of thiamine deficiency would have an adverse effect on the course of heart failure. Routine supplementation of heart failure patients with a high potency B-complex vitamin would therefore be prudent.


Case report: parenteral magnesium for end-stage cardiomyopathy

A 57-year-old man came to see me from several hundred miles away with a 15-year history of slowly progressive dilated cardiomyopathy. He was still functioning relatively well, although he did experience shortness of breath after moderate exertion. The patient was advised to take various nutrients that support cardiovascular function, including coenzyme Q10, magnesium, and L-carnitine, to continue seeing his cardiologist, and to check in with me periodically by phone. He was also advised to take a series of weekly intramuscular magnesium injections, but he was unable to find a practitioner in his town willing to administer them.

Two years later, I received a call from the man's wife. Her husband was in the final stage of his disease. He was in the hospital on a continuous dobutamine drip, which was needed to him alive. The cardiologist's plan was to give the patient time to get his affairs in order, and then to discontinue the dobutamine and allow the patient to die. His wife asked if there was anything else that could be done. I suggested that he be given one gram of magnesium sulfate intramuscularly, daily for several days.

Within ten minutes of receiving the first injection, the patient converted from atrial fibrillation to normal sinus rhythm, and he appeared to be improved overall. He did revert to atrial fibrillation again shortly thereafter, but the series of magnesium injections had a dramatic beneficial effect. He was successfully weaned from the dobutamine drip and later discharged from the hospital.

He continued to receive magnesium injections at home every fourth day. If more than four days elapsed between injections, his condition would deteriorate. Although he was not able to do much more than walk slowly around the house, magnesium injections extended his life by about two years.

Comment: One can only guess how much more the magnesium injections might have helped this man if they had started earlier in the course of his disease.

"Fever of unknown origin" caused by food allergy

Between the ages of 21 and 30 months, a girl was seen by 16 doctors for fever of unknown origin and C-reactive protein concentrations of 100-200 150 mg/L (normal, less than 5 mg/L). Elimination of dairy products and subsequent challenge demonstrated that the fever was due to cow's milk allergy. On a milk-free diet, the C-reactive protein level normalized within one week, and no further fevers occurred.

Comment: Fever has been mentioned repeatedly over the years as a potential manifestation of food allergy. As early as 1937, one practitioner described three patients with constant fever for long periods of time (up to eight years in one case) that resolved after identification and avoidance of allergenic foods. Despite these reports, allergy is not mentioned as a potential cause of "fever of unknown origin" in standard texts.


Folic acid prevents age-related hearing loss

Seven hundred twenty-eight older men and women (mean age, 60 years) residing in the Netherlands who had plasma total homocysteine concentrations of 13 micromol/L or greater, serum vitamin B12 concentrations of 200 pmol/L or greater, and no pathologic ear conditions were randomly assigned to receive, in double-blind fashion, 800 mcg/day of folic acid or placebo for three years. After three years, the mean threshold for hearing of low frequencies increased by 1.0 dB in the folic acid group and by 1.7 dB in the placebo group (p = 0.02 for the difference in the change between groups). Folic acid supplementation did not affect the decline in hearing of high frequencies.

Comment: Low folate status has been associated with poor hearing. In the new study, folic acid supplementation slowed age-related decline in hearing of the speech frequencies. The study was conducted in a country in which food is not fortified with folic acid, so it is not clear whether similar results would be seen in the United States, where grains are fortified with folic acid. Nor is it clear whether people with normal homocysteine levels would obtain the same benefit from folic acid. Folic acid supplementation should be combined with vitamin B12, because these nutrients work together in the body and administering a large dose of one might promote a deficiency of the other.


Eggs are a good source of lutein and zeaxanthin

Twenty-four female volunteers (aged 24-59 years) were assigned to consume six eggs per week or a placebo pill, while continuing their normal diet, for 12 weeks. The eggs were obtained either from a local supermarket (EGG 1) or from an organic farm (EGG 2). The lutein and zeaxanthin content of the eggs varied considerably, but the means were higher in EGG 2 than in EGG 1 (964 vs. 331 mcg of lutein + zeaxanthin per yolk). Serum zeaxanthin, but not serum lutein, increased in both the EGG 1 (p = 0.04) and EGG 2 (p = 0.01) groups. Macular pigment optical density increased in both the EGG 1 (p = 0.001) and EGG 2 (p < 0.05) groups. The higher-carotenoid eggs were not more effective at increasing macular pigment optical density than the lower-carotenoid eggs.

Comment: The macular pigment, which consists largely of the carotenoids lutein and zeaxanthin, appears to protect against the development of age-related macular degeneration, apparently by filtering the phototoxic blue-light portion of the spectrum. Previous studies have shown that people with a thicker macular pigment (as measured by macular pigment optical density) have a lower risk of developing macular degeneration. Although the content of lutein + zeaxanthin in eggs is relatively modest relative to other sources such as spinach, their bioavailability to the retina appears to be high, as demonstrated in this study by an increase in the macular pigment optical density.

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